REMARKS

With this Amendment, Applicants have canceled Claims 2, 15, 24, 26-29, 31-32, 34-38 and 41-49 without prejudice. Claims 1, 3-14, 16-23, 25, 33 and 39 have been amended. After entry of the instant amendment, Claims 1, 3-14, 16-23, 25, 30, 33, 39-40 and 50-52 are pending. A marked up copy of the amended claims is attached at Exhibit A. For the Examiner's convenience, a clean copy of all pending claims is attached at Exhibit B.

Applicants reserve the right to prosecute any canceled subject matter in one or more continuation, divisional or continuation-in-part applications.

I. THE AMENDMENT OF THE CLAIMS

In general, the claims have been amended to recite D-enantiomeric ApoA-I agonist compounds, lipid complexes, pharmaceutical compositions and methods of use thereof. Support for amended Claims 1, 3-14, 16-23, 25, 30 and 39 may be found, for example, in Claims 1, 3-14, 16-23, 25, 33 and 39 as originally filed and in the specification, for example, at page 44, lines 15 to 29.

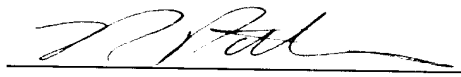
CONCLUSION

Applicants submit that Claims 1, 3-14, 16-23, 25, 30, 33, 39-40 and 50-52 satisfy all the criteria for patentability and are in condition for allowance. An early indication of the same is therefore kindly solicited.

Pursuant to 37 C.F.R. §1.136 (a)(3), the Commissioner is authorized to charge all required fees, fees under 37 C.F.R. §1.17 and all required extension of time fees, or credit any overpayment, to Pennie & Edmonds LLP, U.S. Deposit Account No. 16-1150 (Order No. 9196-019-999). A Fee Transmittal Sheet is enclosed (in duplicate) for accounting purposes.

Respectfully submitted,

Date: March 15, 2002



42,983
Rahul Pathak (Reg No.)
for Laura A. Coruzzi (Reg. No. 30,742)
PENNIE & EDMONDS LLP
1155 Avenue of the Americas
New York, New York 10036-2711
(212) 790-9090

EXHIBIT A

Claim Amendment: Marked Up Copy

1. (Amended) An ApoA-I agonist compound comprising:

(i) [a 14] an 18 to 22-residue D-enantiomeric peptide or peptide analogue which forms an amphipathic α-helix in the presence of lipids and which comprises [the structural] formula (I):

Z_1-X_1-X_2-X_3-X_4-X_5-X_6-X_7-X_8-X_9-X_{10}-X_{11}-X_{12}-X_{13}-X_{14}-X_{15}-X_{16}-X_{17}-X_{18}-Z_2

X_1 is [Pro (P),] D-Ala [(A)] (a), Gly (G), D-Asn [(N)] (n), D-Gln [(Q)] (q) or D-Pro (p);

X_2 is [an] a D-enantiomeric aliphatic [amino acid] residue;

X_3 is D-Leu [(L)] (l);

X_4 is [an] a D-enantiomeric acidic [amino acid] residue;

X_5 is D-Leu [(L)] (l) or D-Phe [(F)] (f);

X_6 is D-Leu [(L)] (l) or D-Phe [(F)] (f);

X_7 is a D-enantiomeric basic [amino acid] residue;

X_8 is [an] a D-enantiomeric acidic [amino acid] residue;

X_9 is D-Leu [(L)] (l) or D-Trp [(W)] (w);

X_{10} is D-Leu [(L)] (l) or D-Trp [(W)] (w);

X_{11} is [an] a D-enantiomeric acidic [amino acid] residue or D-Asn [(N)] (n);

X_{12} is [an] a D-enantiomeric acidic [amino acid] residue;

X_{13} is D-Leu [(L)] (l), D-Trp [(W)] (w) or D-Phe [(F)] (f);

X_{14} is a D-enantiomeric basic [amino acid] residue or D-Leu [(L)] (l);

X_{15} is D-Gln [(Q)] (q) or D-Asn [(N)] (n);

X_{16} is a D-enantiomeric basic [amino acid] residue;

X_{17} is D-Leu [(L)] (l);

X_{18} is a D-enantiomeric basic [amino acid] residue;

Z_1 is [H_2N- or RC(O)NH-] R_2N-, or RC(O)NR-;

Z_2 is -C(O)NRR, -C(O)OR or -C(O)OH or a salt thereof;

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl, (C_5-C_{20}) aryl, (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl or 6-26 membered alkheteroaryl or a 1 to [4] 7-residue peptide or peptide analogue in which one or more bonds between residues 1 through 7 are independently a substituted amide, an isostere of an amide or an amide mimetic; and

each " - " between residues [X_n] X_1 through X_{18} independently designates an amide linkage, a substituted amide linkage, an isostere of an amide or an amide mimetic; or

(ii) a 14 to 21-deleted [from of structural] D-enantiomeric peptide or peptide analogue according to formula (I) in which at least one and up to eight of residues X_1, X_2, X_3, X_4, X_5, X_6, X_7, X_8, X_9, X_{10}, X_{11}, X_{12}, X_{13}, X_{14}, X_{15}, X_{16}, X_{17} and X_{18} are optionally deleted; or

(iii) an 18-22-altered [form of structural] D-enantiomeric peptide or peptide analogue according to formula (I) in which at least one of residues X_1, X_2, X_3, X_4, X_5, X_6, X_7, X_8, X_9, X_{10}, X_{11}, X_{12}, X_{13}, X_{14}, X_{15}, X_{16}, X_{17} or X_{18} is conservatively substituted with another D-enantiomeric residue.

3. (Amended) The ApoA-I agonist compound of Claim 1 which is the altered [form of structural] D-enantiomeric peptide or peptide analogue according to formula (I).

4. (Amended) The ApoA-I agonist compound of Claim 3 in which the D-enantiomeric hydrophobic residues are fixed according to [structural] formula (I) and at least one non-fixed residue is conservatively substituted with another D-enantiomeric residue.

5. (Amended) The ApoA-I agonist compound of Claim 4 in which:

X_1 is [Pro (P)] D-Pro (p), Gly (G), D-Asn [(N)] (n) or D-Ala [(A)] (a);

X_2 is D-Ala [(A)] (a), D-Leu [(L)] (l) or D-Val [(V)] (v);

X_3 is D-Leu [(L)] (l);

X_5 is D-Leu [(L)] (l) or D-Phe [(F)] (f);

X_6 is D-Leu [(L)] (l) or D-Phe [(F)] (f);

X_9 is D-Leu [(L)] (l) or D-Trp [(W)] (w);

X_{10} is D-Leu [(L)] (l) or D-Trp [(W)] (w);

X_{13} is D-Leu [(L)] (l), D-Trp [(W)] (w) or D-Phe [(F)](f);

X_{17} is D-Leu [(L)] (l); and

at least one of X_4, X_7, X_8, X_{11}, X_{12}, X_{14}, X_{15}, X_{16} and X_{18} is conservatively substituted with another D-enantiomeric residue.

6. (Amended) The ApoA-I agonist compound of Claim 3 in which the D-enantiomeric hydrophilic residues are fixed according to [structural] formula (I) and at least one non-fixed residue is conservatively substituted with another D-enantiomeric residue.

7. (Amended) The ApoA-I agonist compound of Claim 6 in which:

X_4 is D-Asp [(D)] (d) or D-Glu [(E)] (e);

X_7 is D-Arg [(R)] (r), D-Lys [(K)] (k) or D-Orn;

X_8 is D-Asp [(D)] (d) or D-Glu [(E)] (e);

X_{11} is D-Asn [(N)] (n) or D-Glu [(E)] (e);

X_{12} is D-Glu [(E)] (e);

X_{14} is D-Lys [(K)] (k), D-Arg [(R)] (r) or D-Orn;

X_{15} is D-Gln [(Q)] (q) or D-Asn [(N)] (n);

X_{16} is D-Lys [(K)] (k), D-Arg [(R)] (r) or D-Orn;

X_{18} is D-Asn [(N)] (n) or D-Gln [(Q)] (q); and

at least one of X_1, X_2, X_3, X_5, X_6, X_9, X_{10}, X_{13} and X_{17} is conservatively substituted with another D-enantiomeric residue.

8. (Amended) The ApoA-I agonist compound of Claim 6 in which X_3 is D-Leu [(L)] (l), X_6 is D-Phe [(F)] (f), X_9 is D-Leu [(L)] (l) or D-Trp [(W)] (w), X_{10} is D-Leu [(L)] (l) or D-

Trp [(W)] (w) and at least one of X_1, X_2, X_5, X_{13} and X_{17} is conservatively substituted with another D-enantiomeric residue.

9. (Amended) The ApoA-I agonist compound of Claim 5 or 7 in which the substituting D-enantiomeric residue is classified within the same sub-category as the substituted D-enantiomeric residue.

10. (Amended) The ApoA-I agonist compound of Claim 1 which is the deleted [form of structural] D-enantiomeric peptide or peptide analogue according to formula (I).

11. (Amended) The ApoA-I agonist compound of Claim 10 in which one or two helical [turn] turns of the D-enantiomeric peptide or peptide analogue is optionally deleted.

12. (Amended) The ApoA-I agonist compound of Claim 1 which is an 18-residue D-enantiomeric peptide or peptide analogue according to [of structural] formula (I).

13. (Amended) The ApoA-I agonist compound of Claim 12 in which
the "-" between residues designates -C(O)NH-;
Z_1 is H_2N-; and
Z_2 is -C(O)OH or a salt thereof.

14. (Amended) The ApoA-I agonist compound of Claim 13, in which:
X_1 is [Pro (P),] D-Ala [(A)] (a), Gly (G), D-Asn [(N)] (n) or D-Pro (p);
X_2 is D-Ala [(A)] (a), D-Val [(V)] (v), or D-Leu [(L)] (l);
X_3 is D-Leu [(L)] (l);
X_4 is D-Asp [(D)] (d) or D-Glu [(E)] (e);
X_5 is D-Leu [(L)] (l) or D-Phe [(F)] (f);
X_6 is D-Leu [(L)] (l) or D-Phe [(F)] (f);
X_7 is D-Arg [(R)] (r), D-Lys [(D)] (d) or D-Orn;

X_8 is D-Asp [(D)] (d) or D-Glu [(E)] (e);

X_9 is D-Leu [(L)] (l) or D-Trp [(W)] (w);

X_{10} is D-Leu [(L)] (l) or D-Trp [(W)] (w);

X_{11} is D-Glu [(E)] (e) or D-Asn [(N)] (n);

X_{12} is D-Glu [(E)] (e);

X_{13} is D-Leu [(L)] (l), D-Trp [(W)] (w) or D-Phe [(F)] (f);

X_{14} is D-Arg [(R)] (r), D-Lys [(K)] (k) or D-Orn;

X_{15} is D-Gln [(Q)] (q) or D-Asn [(N)] (n);

X_{16} is D-Arg [(R)] (r), D-Lys [(K)] (k) or D-Orn;

X_{17} is D-Leu [(L)] (l); and

X_{18} is D-Arg [(R)] (r), D-Lys [(D)] (d) or D-Orn.

16. (Amended) A multimeric ApoA-I agonist compound [which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural] which comprises formula (II):

(II) $$HH\{LL_m\text{-}HH\}_nLL_m\text{-}HH$$

or a pharmaceutically acceptable salt thereof, wherein:

each m is independently an integer from 0 to 1;

n is an integer from 0 to 10;

each "HH" is independently a peptide or peptide analogue according to Claim 1;

each "LL" is independently a bifunctional linker; and

each " - " independently designates a covalent linkage.

17. (Amended) A multimeric ApoA-I agonist compound [which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and] which comprises [has the structural] formula (III):

(III) $X\text{-}N_{ya}\text{-}X_{(ya-1)}\text{-}(\text{-}N_{yb}\text{-}X_{(yb-1)})_p$

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently $HH\text{-}(\text{-}LL_m\text{—}HH\text{-})_nLL_m\text{—}HH$;

each HH is independently a [core] peptide [of structure (I) or an analogue or mutated, truncated, internally deleted or extended form thereof as described herein] <u>or peptide analogue according to Claim 1</u>;

each LL is independently a bifunctional linker;

each m is independently an integer from 0 to 1;

each n is independently an integer from 0 to 8;

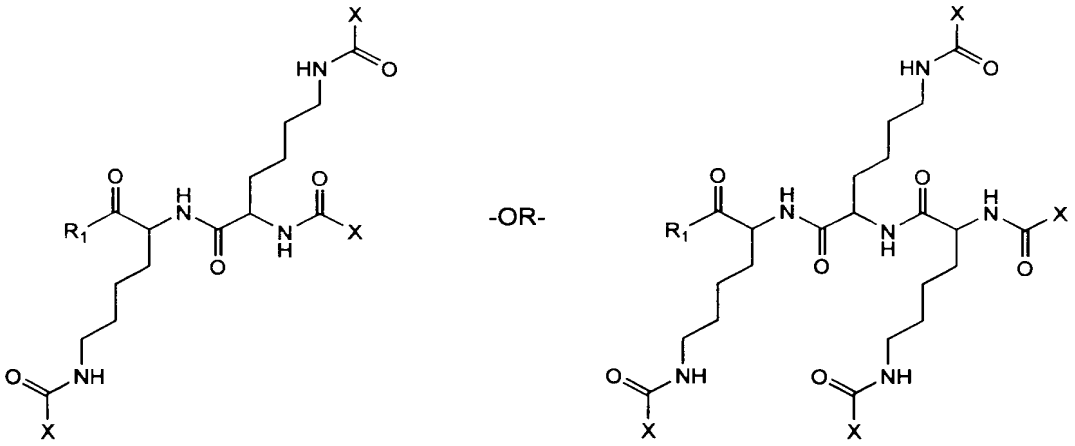
N_{ya} and N_{yb} are each independently a multifunctional linking moiety where y_a and y_b represent the number of functional groups on N_{ya} and N_{yb}, respectively;

each y_a or y_b is independently an integer from 3 to 8;

p is an integer from 0 to 7; and

each "—" independently designates a covalent bond.

18. (Amended) A multimeric ApoA-I agonist compound which [exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural] comprises formula (IV) or (V):



(IV) (V)

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently HH-(-LL_m—HH-)-$_n$$LL_m$—HH;

each HH is independently a peptide or peptide analogue according to Claim 1;

each LL is independently a bifunctional linker;

each n is independently an integer from 0 to 1;

each m is independently an integer from 0 to 8;

R_1 is -OR or -NRR; and

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl[;], (C_5-C_{20}) aryl, (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl or 6-26 membered alkheteroaryl.

19. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which the bifunctional linker is cleavable.

20. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which n is 0.

21. (Amended) The multimeric ApoA-I agonist compound of Claim 20 in which m is 0.

22. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which each HH is independently a peptide or peptide analogue according to Claim [13] 3.

23. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which each HH is independently a peptide or peptide analogue according to Claim [14] 10.

25. (Amended) An ApoA-I agonist-lipid complex comprising an ApoA-I agonist and a lipid, wherein the ApoA-I agonist is a peptide or peptide analogue according to Claim 1, a multimeric ApoA-I agonist compound according to Claim 16, a multimeric ApoA-I agonist compound according to Claim 17, or a multimeric ApoA-I agonist compound according to Claim 18.

33. (Amended) A pharmaceutical composition comprising an ApoA-I agonist and a pharmaceutically acceptable carrier, excipient or diluent, wherein the ApoA-I agonist is [a peptide or peptide analogue according to Claim 1, a multimeric ApoA-I agonist according to Claim 16, a multimeric ApoA-I agonist according to Claim17, or a multimeric ApoA-I agonist according to Claim18] in the form of an ApoA-I agonist-lipid complex, said complex comprising an ApoA-I agonist compound and a lipid.

39. (Amended) The pharmaceutical composition of Claim [38] 33, [in which the ApoA-I agonist-lipid complex] which is in form of a lyophilized powder.

EXHIBIT B

Claim Amendment: Pending Claims After Entry of Instant Amendment

1. (Amended) An ApoA-I agonist compound comprising:

(i) an 18 to 22-residue D-enantiomeric peptide or peptide analogue which forms an amphipathic α-helix in the presence of lipids and which comprises formula (I):

Z_1-X_1-X_2-X_3-X_4-X_5-X_6-X_7-X_8-X_9-X_{10}-X_{11}-X_{12}-X_{13}-X_{14}-X_{15}-X_{16}-X_{17}-X_{18}-Z_2

X_1 is D-Ala (a), Gly (G), D-Asn (n), D-Gln (q) or D-Pro (p);

X_2 is a D-enantiomeric aliphatic residue;

X_3 is D-Leu (l);

X_4 is a D-enantiomeric acidic residue;

X_5 is D-Leu (l) or D-Phe (f);

X_6 is D-Leu (l) or D-Phe (f);

X_7 is a D-enantiomeric basic residue;

X_8 is a D-enantiomeric acidic residue;

X_9 is D-Leu (l) or D-Trp (w);

X_{10} is D-Leu (l) or D-Trp (w);

X_{11} is a D-enantiomeric acidic residue or D-Asn (n);

X_{12} is a D-enantiomeric acidic residue;

X_{13} is D-Leu (l), D-Trp (w) or D-Phe (f);

X_{14} is a D-enantiomeric basic residue or D-Leu (l);

X_{15} is D-Gln (q) or D-Asn (n);

X_{16} is a D-enantiomeric basic residue;

X_{17} is D-Leu (l);

X_{18} is a D-enantiomeric basic residue;

Z_1 is R_2N-, or RC(O)NR-;

Z_2 is -C(O)NRR, -C(O)OR or -C(O)OH or a salt thereof;

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl, (C_5-C_{20}) aryl, (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl or 6-26 membered alkheteroaryl or a 1 to 7-residue peptide or peptide analogue in which one or more bonds between residues 1 through 7 are independently a substituted amide, an isostere of an amide or an amide mimetic; and

each " - " between residues X_1 through X_{18} independently designates an amide linkage, a substituted amide linkage, an isostere of an amide or an amide mimetic; or

(ii) a 14 to 21-deleted D-enantiomeric peptide or peptide analogue according to formula (I) in which at least one and up to eight of residues X_1, X_2, X_3, X_4, X_5, X_6, X_7, X_8, X_9, X_{10}, X_{11}, X_{12}, X_{13}, X_{14}, X_{15}, X_{16}, X_{17} and X_{18} are optionally deleted; or

(iii) an 18 to 21-altered D-enantiomeric peptide or peptide analogue according to formula (I) in which at least one of residues X_1 , X_2 , X_3 , X_4 , X_5 , X_6 , X_7 , X_8 , X_9 , X_{10} , X_{11} , X_{12} , X_{13} , X_{14} , X_{15} , X_{16} , X_{17} and X_{18} is conservatively substituted with another D-enantiomeric residue.

3. (Amended) The ApoA-I agonist compound of Claim 1 which is the altered D-enantiomeric peptide or peptide analogue according to formula (I).

4. (Amended) The ApoA-I agonist compound of Claim 3 in which the D-enantiomeric hydrophobic residues are fixed according to formula (I) and at least one non-fixed residue is conservatively substituted with another D-enantiomeric residue.

5. (Amended) The ApoA-I agonist compound of Claim 4 in which:

X_1 is D-Pro (p), Gly (G), D-Asn (n) or D-Ala (a);

X_2 is D-Ala (a), D-Leu (l) or D-Val (v);

X_3 is D-Leu (l);

X_5 is D-Leu (l) or D-Phe (f);

X_6 is D-Leu (l) or D-Phe (f);

X_9 is D-Leu (l) or D-Trp (w);

X_{10} is D-Leu (l) or D-Trp (w);

X_{13} is D-Leu (l), D-Trp (w) or D-Phe (f);

X_{17} is D-Leu (l); and

at least one of X_4, X_7, X_8, X_{11}, X_{12}, X_{14}, X_{15}, X_{16} and X_{18} is conservatively substituted with another D-enantiomeric residue.

6. (Amended) The ApoA-I agonist compound of Claim 3 in which the D-enantiomeric hydrophilic residues are fixed according to formula (I) and at least one non-fixed residue is conservatively substituted with another D-enantiomeric residue.

7. (Amended) The ApoA-I agonist compound of Claim 6 in which:

X_4 is D-Asp (d) or D-Glu (e);

X_7 is D-Arg (r), D-Lys (k) or D-Orn;

X_8 is D-Asp (d) or D-Glu (e);

X_{11} is D-Asn (n) or D-Glu (e);

X_{12} is D-Glu (e);

X_{14} is D-Lys (k), D-Arg (r) or D-Orn;

X_{15} is D-Gln (q) or D-Asn (n);

X_{16} is D-Lys (k), D-Arg (r) or D-Orn;

X_{18} is D-Asn (n) or D-Gln (q); and

at least one of X_1, X_2, X_3, X_5, X_6, X_9, X_{10}, X_{13} and X_{17} is conservatively substituted with another D-enantiomeric residue.

8. (Amended) The ApoA-I agonist compound of Claim 6 in which X_3 is D-Leu (l), X_6 is Phe (f), X_9 is D-Leu (l) or D-Trp (w), X_{10} is D-Leu (l) or D-Trp (w) and at least one of X_1, X_2, X_5, X_{13} and X_{17} is conservatively substituted with another D-enantiomeric residue.

9. (Amended) The ApoA-I agonist compound of Claim 5 or 7 in which the substituting D-enantiomeric residue is classified within the same subcategory as the substituted D-enantiomeric residue.

10. (Amended) The ApoA-I agonist compound of Claim 1 which is the deleted D-enantiomeric peptide or peptide analogue according to formula (I).

11. (Amended) The ApoA-I agonist compound of Claim 10 in which one or two helical turns of the D-enantiomeric peptide or peptide analogue is optionally deleted.

12. (Amended) The ApoA-I agonist compound of Claim 1 which is an 18-residue D-enantiomeric peptide or peptide analogue according to formula (I).

13. (Amended) The ApoA-I agonist compound of Claim 12 in which
the "-" between residue designates -C(O)NH-;
Z_1 is H_2N-; and
Z_2 is -C(O)OH or a salt thereof.

14. (Amended) The ApoA-I agonist compound of Claim 13, in which;
X_1 is D-Ala (a), Gly (G), D-Asn (n) or D-Pro (p);
X_2 is D-Ala (a), D-Val (v), or D-Leu (l);
X_3 is D-Leu (l);
X_4 is D-Asp (d) or D- Glu (e);
X_5 is D-Leu (l) or D-Phe (f);
X_6 is D-Leu (l) or D-Phe (f);
X_7 is D-Arg (r), D-Lys (d) or D-Orn;
X_8 is D-Asp (d) or D-Glu (e);
X_9 is D-Leu (l) or D-Trp (w);
X_{10} is D-Leu (l) or D-Trp (w);

X_{11} is D-Glu (e) or D-Asn (n);

X_{12} is D-Glu (e);

X_{13} D-Leu (l), D-Trp (w) or D-Phe (f);

X_{14} is D-Arg (r), D-Lys (k) or D-Orn;

X_{15} is D-Gln (q) or D-Asn (n);

X_{16} is D-Arg (r), D-Lys (k) or D-Orn;

X_{17} is D-Leu (l); and

X_{18} is D-Arg (r), D-Lys (d) or D-Orn.

16. (Amended) A multimeric ApoA-I agonist compound which comprises formula (II):

(II) $HH\{LL_m\text{-}HH\}_nLL_m\text{-}HH$

or a pharmaceutically acceptable salt thereof, wherein:

each m is independently an integer from 0 to 1;

n is an integer from 0 to 10;

each "HH" is independently a peptide or peptide analogue according to Claim 1;

each "LL" is independently a bifunctional linker; and

each " - " independently designates a covalent linkage.

17. (Amended) A multimeric ApoA-I agonist compound which comprises formula (III):

(III) $X\text{-}N_{ya}\text{-}X_{(ya-1)}\text{-}(\text{-}N_{yb}\text{-}X_{(yb-1)})_p$

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently $HH\text{-}(\text{-}LL_m\text{—}HH\text{-})_nLL_m\text{—}HH$;

each HH is independently a peptide or peptide analogue according to Claim 1;

each LL is independently a bifunctional linker;

each m is independently an integer from 0 to 1;

each n is independently an integer from 0 to 8;

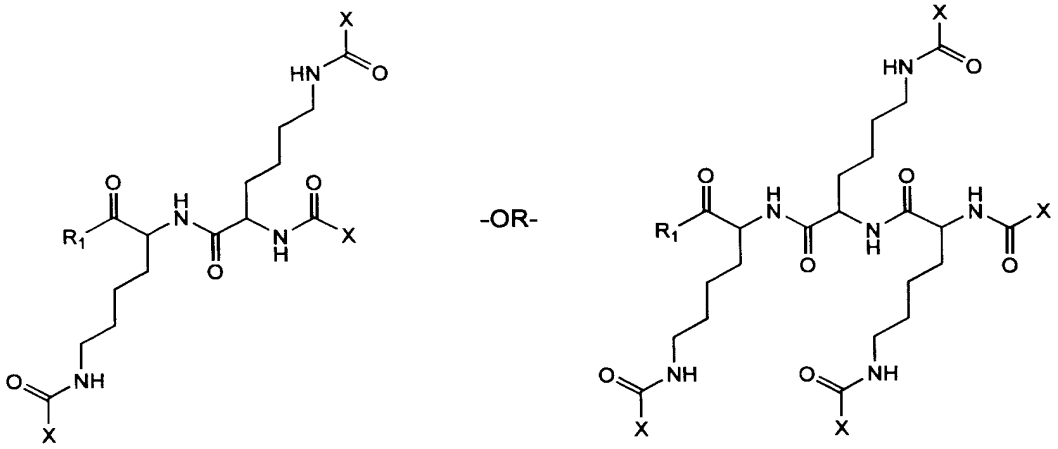
N_{ya} and N_{yb} are each independently a multifunctional linking moiety where y_a and y_b represent the number of functional groups on N_{ya} and N_{yb}, respectively;

each y_a or y_b is independently an integer from 3 to 8;

p is an integer from 0 to 7; and

each "—" independently designates a covalent bond.

18. (Amended) A multimeric ApoA-I agonist compound which comprises formula (IV) or (V):



(IV) (V)

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently HH-(-LL_m—HH-)-$_n LL_m$—HH;

each HH is independently a peptide or peptide analogue according to Claim 1;

each LL is independently a bifunctional linker;

each n is independently an integer from 0 to 1;

each m is independently an integer from 0 to 8;

R_1 is -OR or -NRR; and

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl, (C_5-C_{20}) aryl, (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl or 6-26 membered alkheteroaryl.

19. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which the bifunctional linker is cleavable.

20. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which n is 0.

21. (Amended) The multimeric ApoA-I agonist compound of Claim 20 in which m is 0.

22. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which each HH is independently a peptide or peptide analogue according to Claim 3.

23. (Amended) The multimeric ApoA-I agonist compound of Claim 16, 17 or 18 in which each HH is independently a peptide or peptide analogue according to Claim 10.

25. (Amended) An ApoA-I agonist-lipid complex comprising an ApoA-I agonist and a lipid, wherein the ApoA-I agonist is a peptide or peptide analogue according to Claim 1, a multimeric ApoA-I agonist compound according to Claim 16, a multimeric ApoA-I agonist compound according to Claim 17, or a multimeric ApoA-I agonist compound according to Claim 18.

30. The ApoA-I agonist-lipid complex of Claim 25 in which the lipid is sphingomyelin.

33. (Amended) A pharmaceutical composition comprising an ApoA-I agonist and a pharmaceutically acceptable carrier, excipient or diluent, wherein the ApoA-I agonist is in the form of an ApoA-I agonist-lipid complex, said complex comprising an ApoA-I agonist compound and a lipid.

39. (Amended) The pharmaceutical composition of Claim 33, which is in the form of a lyophilized powder.

40. A method of treating a subject suffering from a disorder associated with dyslipidemia, said method comprising the step of administering to the subject an effective amount of the ApoA-I agonist of Claim 1.

50. A method of treating a subject suffering from septic shock, said method comprising the step of administering to the subject an effective amount of the ApoA-I agonist of Claim 1.

51. The method of Claim 40 or 50 in which said subject is a human.

52. The method of Claim 40 or 50 in which about 0.5 mg/kg to about 100 mg/kg ApoA-I agonist is administered to said subject.